



A Severe Hypothyroidism Leading to Reversible Dilated Cardiomyopathy

Eljazouli Ali ^{a*}, Nachid Mohammed ^a, Salim Arous ^a,
Bennouna G. Hali ^a and Habbal Rachida ^a

^a Cardiology Division, Ibn Rochd University Hospital, Casablanca 20250, Morocco.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

Open Peer Review History:

This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: <https://www.sdiarticle5.com/review-history/100424>

Case Study

Received: 18/03/2023

Accepted: 23/05/2023

Published: 03/06/2023

ABSTRACT

DCM, a heart condition causing muscle weakness and enlargement, is often associated with hypothyroidism. A 35-year-old woman with no prior medical history was diagnosed with DCM due to hypothyroidism after experiencing chest pain, fatigue, and shortness of breath. Echocardiography revealed severe dysfunction in her left ventricle, with an ejection fraction of only 25%. Further examination showed low levels of free thyroxine (FT4) and elevated levels of thyroid-stimulating hormone (TSH). The patient's cardiac function significantly improved over several months following the initiation of thyroid hormone replacement therapy. The case highlights the importance of timely identification and management of hypothyroidism-induced DCM to prevent further cardiac complications.

Keywords: *Hypothyroidism; dilated left ventricle; reversible cardiopathy.*

*Corresponding author: E-mail: eljazouliali@hotmail.fr;

1. INTRODUCTION

Hypothyroidism is a prevalent endocrine disorder affecting 5-15% of the population, stemming from insufficient thyroid hormone production [1]. This condition leads to a range of clinical symptoms, including cardiovascular disease. Dilated cardiomyopathy (DCM) is a rare but serious complication of hypothyroidism, and its pathophysiology is still not entirely understood. It is believed that hypothyroidism-induced DCM may be related to impaired cardiac contractility and increased peripheral vascular resistance [2]. Timely diagnosis and treatment are essential to prevent further cardiac complications and improve patient outcomes. A recent case report details the effective treatment of a patient with hypothyroidism-induced DCM through thyroid hormone replacement therapy.

2. LEARNING OBJECTIVES

Recognize the clinical manifestations and diagnostic criteria for severe hypothyroidism and dilated cardiomyopathy.

Explore the potential complications and risks associated with severe hypothyroidism-induced dilated cardiomyopathy.

Understand the prognosis and long-term outcomes associated with severe hypothyroidism-induced dilated cardiomyopathy

3. CASE REPORT

A 35 years old female, arrived at our Emergency Department with various ailments. Among her complaints were shortness of breath upon exertion and swelling throughout her body. Over the past fortnight, her dyspnea had worsened,

even with minimal effort, and her abdomen had become increasingly distended. She had no personal or familial medical history of DCM and had not used any drugs, alcohol, or toxic substances.

The patient's pulse was steady at 70 beats per minute, with normal volume. Her blood pressure measured at 120/60 mm of Hg and her respiratory rate was 23 c per minute. Along with reported voice hoarseness that had persisted for two years, the patient presented with bilateral pitting pedal edema. Furthermore, bulging neck veins and bilateral basal crepitations in her chest were observed during examination.

The electrocardiogram showed a sinus rhythm with complete left bundle branch block (Fig. 1). Chest X-ray revealed grade III cardiomegaly.

Echocardiography showed a dilated left ventricle with severe systolic dysfunction and an ejection fraction of 25%. There was also moderate mitral regurgitation and tricuspid regurgitation. The pulmonary artery pressure was elevated at 45 mmHg.

Laboratory investigations were carried out to assess the patient's health status. The complete blood count (CBC), renal function test (RFT), liver function test (LFT), total serum protein, and albumin levels were all within the normal range. However, the thyroid function test revealed elevated levels of thyroid-stimulating hormone (>40mUI/l) (normal range: 0.27-4.20 mIU/L) and decreased T3 and T4 levels (<0.4 nmol/l). Additional studies were performed to validate these findings, which showed that the antimicrosome antibody and antithyroglobulin antibody were both negative [3].

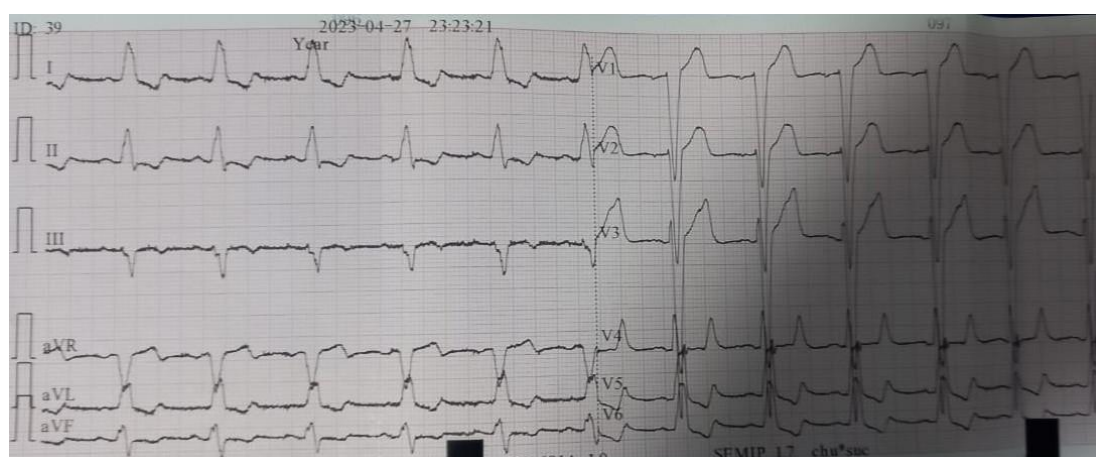


Fig. 1. Electrocardiogram showing complete left bundle branch block

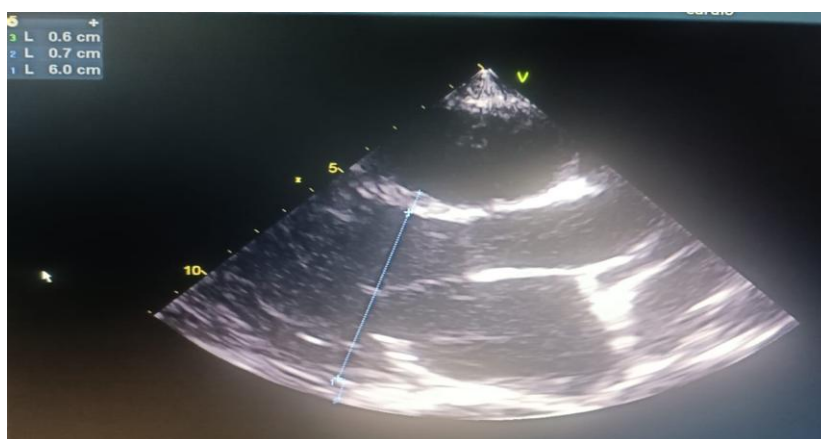


Fig. 2. TTE showing dilated left ventricle

To explore the potential for autoimmune pathology, immunofluorescence was used to conduct antinuclear antibody testing. However, the results did not show any significant findings.

These findings were consistent with severe hypothyroidism. The patient was started on levothyroxine 50mcg daily, and his cardiac medications were optimized.

After several months of thyroid hormone replacement therapy, the patient's cardiac function improved significantly. A follow-up echocardiogram showed an ejection fraction of 55%, and there was resolution of the mitral and tricuspid regurgitation. The patient's symptoms of shortness of breath and fatigue also resolved.

4. DISCUSSION

Hypothyroidism, an endocrine disorder, is prevalent among 5-15% of the population, with varying clinical symptoms that are often nonspecific. However, the correlation between hypothyroidism and cardiovascular disease is well-established. Although rare, the condition can lead to serious complications, such as hypothyroidism-induced DCM, with severe consequences for morbidity and mortality.

By utilizing both genomic and nongenomic pathways, Triiodothyronine (T3) has a direct impact on the cardiovascular system. Genomic pathways involve T3 binding to nuclear receptors, which then regulate the expression of certain proteins via activation or repression of gene expression. Nongenomic pathways include adjustments to the myocyte plasma membrane through alterations in potassium, sodium, and calcium ion channels, alongside changes in cytoskeleton polymerization [4,5]. Furthermore, the Renin-Angiotensin-Aldosterone System and

catecholamines also play a role in abnormal thyroid states' cardiac effects [5,6].

Documented cases of hypothyroidism-induced DCM with reduced LV systolic function are scarce in literature [7]. The inaugural account of DCM in four hypothyroid patients dates back to 1918, with only a few cases surfacing subsequently. One such case, reported by Bezdahet al., revealed heart failure in a patient with severe hypothyroidism and DCM, but the patient recovered through levothyroxine therapy [8]. They recommended that physicians consider hypothyroidism as a likely cause when diagnosing DCM. Additionally, Ladenson et al. discovered reversible changes in myocardial gene expression in a young male with hypothyroidism-induced DCM [9]. Khochtali et al. also documented two case studies revealing hypothyroidism as a reversible cause of DCM [10].

The underlying mechanism of DCM in hypothyroidism is not fully understood. However, it is thought to be due to impaired cardiac contractility and increased peripheral vascular resistance [2]. The most common echocardiographic findings in hypothyroidism-induced DCM are left ventricular dilatation, systolic dysfunction, and diastolic dysfunction [3]. Treatment with thyroid hormone replacement therapy has been shown to improve cardiac function in patients with hypothyroidism-induced DCM [11]. The early detection and management of hypothyroidism-induced DCM can prevent further cardiac complications and improve patient outcomes [3].

The case presented above is consistent with previous reports of hypothyroidism-induced DCM. The patient had a history of

hypothyroidism and was not compliant with his thyroid hormone replacement therapy. He presented with symptoms of heart failure and was found to have severe left ventricular dysfunction on echocardiography. Elevated TSH and low FT4 levels confirmed the diagnosis of severe hypothyroidism. Thyroid hormone replacement therapy was initiated, and the patient's cardiac function improved significantly over the course of several months [3,4].

This case highlights the importance of regular monitoring and compliance with thyroid hormone replacement therapy in patients with hypothyroidism [3]. Early detection and management of hypothyroidism-induced DCM can prevent further cardiac complications and improve patient outcomes [3].

5. CONCLUSION

This case report highlights the importance of recognizing the cardiac complications of hypothyroidism, including DCM. Patients with hypothyroidism should be regularly monitored for cardiac function and compliance with thyroid hormone replacement therapy. Early detection and management of hypothyroidism-induced DCM can prevent further cardiac complications and improve patient outcomes.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Biondi B, Cooper DS. The clinical significance of subclinical thyroid dysfunction. *Endocrine reviews*. 2008; 29(1):76-131.
2. Klein I, Danzi S. Thyroid disease and the heart. *Circulation*. 2007;116(10):1725-35.
3. Shin DH, Lee MJ, Kim SJ, Park MJ, Kim YJ, Kim KI, Kim CH, Park SM. Dilated cardiomyopathy as the first clinical presentation of hypothyroidism. *Journal of Korean Medical Science*. 2011;26(8):1113-6.
4. Osuna PM, Udovicic M, Sharma MD. Hyperthyroidism and the heart. *Methodist Debaque Cardiovasc J*. 2017;13:60-63. Available:10.14797/mdcj-13-2-60
5. Nabbout LA, Robbins RJ. The cardiovascular effects of hyperthyroidism. *Methodist Debaque Cardiovasc J*. 2010;6:3-8. Available:10.14797/mdcj-6-2-3
6. Vargas-Uricoechea H, Bonelo-Perdomo A, Sierra-Torres CH. Effects of thyroid hormones on the heart. *Clin Investig Arterioscler*. 2014;26:296-309. Available:10.1016/j.arteri.2014.07.003
7. Zondek H. Das myxodemherz. *Munch Med Wochenschr*. 1918;65:1180-3.
8. Bezdah L, Slimène H, Kammoun M, Haddad A, Belhani A. Hypothyroid dilated cardiomyopathy. *Ann Cardiol Angeiol (Paris)*. 2004;53:217-20.
9. Ladenson PW, Sherman SI, Baughman KL, Ray PE, Feldman AM. Reversible alterations in myocardial gene expression in a young man with dilated cardiomyopathy and hypothyroidism. *Proc Natl Acad Sci U S A*. 1992;89:5251-5.
10. Khochtali I, Hamza N, Harzallah O, Hamdi S, Saad J, Golli M, et al. Reversible dilated cardiomyopathy caused by hypothyroidism. *Int Arch Med*. 2011;4:20.
11. Siaplaouras J, Anastasiou-Nana MI, Alexopoulos D, Kanakakis J, Asimakopoulos G, Nanas JN. Dilated cardiomyopathy as the presenting manifestation of hypothyroidism: reversal of clinical and echocardiographic abnormalities with replacement therapy. *Hellenic Journal of Cardiology*. 2005; 46(2):96-9.

© 2023 Ali et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:
The peer review history for this paper can be accessed here:
<https://www.sdiarticle5.com/review-history/100424>